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ABSTRACT

Despite years of concern about the toxic effects of high lead exposure and recent knowledge about the less apparent effects of exposure to low doses of lead, a total of 3 to 4 million children in the United States are still being exposed to concentrations of lead that could compromise their cognitive and social development. This paper discusses: (1) the long-term consequences of early exposure to lead; (2) prenatal exposure to lead; (3) definitions of a toxic dose of lead; and (4) reasons why little is being done to control lead exposure. In discussing the long-term effects of lead exposure, the report examines behavioral problems that often occur in children with exposures to low levels of lead and that may lead to children's failure in school. Data examined indicate that lead exposure is greater than average in groups in which subnormal intelligence, attention disorders, and behavior problems are found at higher than average rates; and that half of the black children living in poverty have elevated blood lead levels. Steps in a plan to prevent childhood lead poisoning, announced by the Centers for Disease Control in 1991, include an increase in the number of childhood lead poisoning prevention programs, effective abatement of leaded homes, and a national surveillance effort. (SM)

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The Poisoning of America's Children:

Lead Exposure, Children's Brains, and the Ability to Learn

National Health/Education Consortium Occasional Paper #6

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The Poisoning of America's Children:

Lead Exposure, Children's Brains, and the Ability to Learn

National Health/Education Consortium
Occasional Paper #6

by

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Executive Summary

Any initiative focused on improving the readiness of American children to learn in school must address one of the worst, yet most preventable environmental threats to youngsters — lead exposure. Despite years of concern about the toxic effects of high-lead exposure and recent knowledge about the less apparent low-dose effects, it is estimated that between three and four million children in the United States are still being exposed to concentrations of lead so dangerous, the cognitive and social development of these children could be compromised.

For a number of years, our government has been criticized for not proactively preventing and remedying the situation. Fortunately, however, recent measures taken by the federal government (including lowering the level of lead considered safe in the blood to 10 µg/dl, requiring a diagnostic lead screening test as part of routine physical examinations for Medicaid eligible children, and passing a bill mandating a range of disclosure and hazard reduction measures in federally subsidized housing), should prove to be helpful in diagnosing lead-poisoned children and serve to remind us of the danger of this environmental insult.

Given the federal government's re-evaluation of the problem of lead poisoning, it is time for all of us to ask: are we doing enough to provide America's children a lead-free, risk-free environment?

In "The Poisoning of America's Children: Lead Exposure, Children's Brains, and the Ability to Learn," Dr. Herbert Needleman of the University of Pittsburgh provides a timely, comprehensive overview of lead poisoning, presenting compelling research that demonstrates the long and short-term effects of low level lead exposure on children's intellectual ability and social adjustment. The National Health/Education Consortium, a coalition of 56 national health and education organizations, offers this article not only to educate, but also to once again remind all those concerned with the future of children, to take action on behalf of them. A synopsis of the paper is presented below.

Although it has long been acknowledged that high doses of lead can cause a number of characteristic health problems including headaches, clumsiness, constipation, vomiting and even death, it was not until the 1970s that research began to seriously investigate cases of low dose exposures that do not present the classic symptoms. Despite the fact that some early studies failed to find low dose effects, modern studies using more sensitive measures and larger samples are providing a growing body of data demonstrating that lead exposure, even at very low doses, has a negative and lasting impact on children's development.

The most recent estimates of the number of lead exposed children are startling. Seven percent of children may have lead levels in the range associated with neurobehavioral deficits. When one considers children living in poverty, estimates of the proportion

of children with unhealthy levels of lead run as high as 25% for white children and 55% for African American children.

The threat of lead exposure does not just begin in early childhood but can begin in the prenatal period of development. Lead can pass through the placenta from the mother to the unborn baby where it has been associated with increased rates of minor malformations.

Particularly alarming is the effect of lead exposure on characteristics important for positive school performance. Research at the University of Pittsburgh, led by Dr. Needleman, demonstrates the short-term and long-term effect of lead exposure. His research conducted on ordinary first and second graders provides compelling evidence that children with elevated lead levels have lower IQ scores, poorer speech and language performance, and are more hyperactive and distractible. Follow-up studies with these children show that by fifth grade, children with elevated levels of lead are more likely to have been retained a grade and to have required special school services. By age 18, these same children are at greater risk of having reading problems and of dropping out of high school.

Despite such compelling evidence, concerted efforts to prevent lead poisoning have been slow to develop in the United States. Dr. Needleman attributes this fact to the difficulty of isolating the effects of lead poisoning. Because behavioral problems associated with low dose lead poisoning have been fairly nonspecific in nature, this hazard has been easier to dismiss than others. For poor people and minorities, for instance, the nonspecific symptoms of lead poisoning have been attributed to other factors such as malnutrition and inadequate prenatal care. The difficulty in pinpointing the effects of lead poisoning highlight the importance of the research conducted to date. We must remind ourselves of the dangers and unnecessity of this environmental insult. Since lead poisoning is primarily due to manmade choices, it is potentially completely preventable. Eliminating leaded gasoline and banning lead in household paint are only the first steps to be taken in a primary prevention campaign. Clean-up work needs to be done as well. There are children with elevated lead levels still living in over three million homes with deteriorated leaded surfaces. The cost/benefit ratio associated with deleading these houses shows the overarching benefit of preventive strategies: for every dollar spent, approximately two dollars can be saved from decreased medical and special educational costs and increased wages associated with higher IQ scores.

Even though the challenge of eliminating all lead poisoning in the United States may be daunting, with the proper commitment and leadership in this endeavor, it is within our means to achieve a safer environment and more promising future for our children.

Introduction

The voluminous literature on developmental failure in childhood largely ignores the effect of neurotoxins on the competence of children's brains. This is tragic: since these neurotoxins are products of human choice and activity, their control offers a ready target for authentic primary prevention.

Lead is the best studied neurotoxin; it was known to be dangerous to the ancient Greeks as early as the second century B.C. Throughout history warnings about its hazards to workers, pregnant women, and children have been raised, and regularly ignored. Knowledge of the pathophysiology, epidemiology and long term consequences of childhood lead exposure has grown rapidly in the last decade. This drew the federal government in 1991 to pronounce lead exposure as the "most serious environmental disease of American children," and the Public Health Service has accordingly shifted its strategy from case finding and treatment to authentic primary prevention.[1]

The most recent prevalence of lead exposure figures, taken from federal epidemiologic studies, startled even experienced workers in the field of lead toxicology. The best estimate is that 7% of all American children may have lead levels in the range associated with neurobehavioral deficits.[2] For poor whites, the prevalence was 25% and for poor black children the rate was 55%.

In the following pages I review recent progress in the toxicology of lead-understanding what it does, its epidemiology-where it is, and then examine what is needed to eliminate the problem permanently. I provide data that indicates that lead exposure may be one of the most important and accessible risk factors in the constellation of assaults on children's brains, and that it may play a significant role in intellectual development and school failure. I also investigate some of the forces that have retarded progress in prevention. Unless these forces are understood and confronted, the strong promise that this disease will be banished to the medical history texts will be forfeited.

1 CDC (1991): *Strategic Plan for the Elimination of Childhood Lead Poisoning.* Center for Disease Control, Department of Health and Human Services, Atlanta, GA.

2 ATSDR (1988): *The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress.* Department of Health and Human Services, Atlanta, GA.

History

Lead toxicity has been known since antiquity. In the second century B.C.[3] Dioscorides, perhaps the first neurotoxicologist, said that "Lead makes the mind give way." Two hundred years ago, Benjamin Franklin wrote of the "dry gripes" (colic) and "dangles" (wrist drop) that affected typesetters and painters.[4] Childhood lead poisoning was first described by a pediatric house officer, A. J. Turner, at the Brisbane Children's Hospital at the end of the 19th century.[5] By painstaking observation, noticing that many children were admitted shortly after they had moved to a different house, Turner and his colleague, J. Lockhart Gibson, concluded that the disease was one of "habitation." [6]

In 1920 the Australian Medical Congress passed a resolution asking for a ban on lead paint in houses. It was fifty years later before the United States followed suit.

Childhood lead poisoning was described in the United States in the early part of the 20th century.[7,8] It was generally believed that there were only two possible outcomes to a case of acute lead toxicity, death or complete recovery. In 1943 Randolph Byers, a pediatric neurologist, had noted that some of the cases referred to him for school failure, were the same children he had earlier seen and treated for acute lead poisoning. With Dr Elizabeth Lord, a psychologist, he followed up on 20 children assumed to be completely recovered from plumbism, and found that 19 had school or behavioral problems.[9] Byers raised for the first time the question of whether undiagnosed lead toxicity was a cause of school failure. This paper opened the modern era of lead toxicology.

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3 Major, RH (1931): Some landmarks in the history of lead poisoning. *Annals Med. His.* 3,218-227.

4 Franklin, B (1818): Pernicious effects of lead. In: *The Works of Ben Franklin.* (Ed: Franklin, B) B.C. Buzby, Philadelphia, 284-286.

5 Turner, AJ (1897): Lead poisoning among Queensland children. *Austr Med Gazette.* 16, 475-479.

6 Gibson, JL (1904): A plea for painted railings and painted walls of rooms as the source of lead poisoning among Queensland children. *Austr Med Gazette.* 23, 149-153.

7 McKhann, CF; Vogt, EC (1926): Lead poisoning in children: with notes on therapy. *Am. J. Dis. Child.* 32, 386-392.

8 Blackfan, KD (1917): Lead poisoning in children with special reference to lead as a cause of convulsions. *Am. J. Med. Sci.* 153, 877-887.

9 Byers, RK; Lord EE (1943): Late effects of lead poisoning on mental development. *Am. J. Dis. Child.* 66, 471-483.

Studies of Asymptomatic Exposure

It was assumed in the 1940s and 50s that lead toxicity occurred only in the presence of clear symptoms of headache, clumsiness, constipation or vomiting. Studies of asymptomatic exposed children began to be published in the early 1970's.[10,11,12] Some showed an effect, some did not.

In 1979, my group, after reviewing current studies of this problem, identified four major design problems: poor markers of exposure, weak measures of outcome, inadequate control of covariates, and selection bias. Studies which classified children by blood lead level could misclassify subjects, since the blood is a short term storage system. Group tests or screening tests are inadequate to measure small changes in function at lesser doses. Many factors affect development, and some could be correlated with lead, and thus confound its effects. Finally, subjects drawn from psychiatric or pediatric clinics may not be representative of the population of children at risk. We designed a study to address these issues.[13] We used as a marker of exposure, dentine lead levels, since these represent past exposure to lead. We gave our subjects, chosen from ordinary first and second grades in Somerville and Chelsea, MA, a large battery of measures. We evaluated psychometric intelligence, attention, language function, and behavior, and controlled for a number of nonlead factors. After control of socioeconomic covariates including mothers' IQs, high lead children had 4 points lower mean IQ scores than their low lead counterparts. They also had inferior scores on measures of speech and language function, and on attention. When teachers rated children on an 11 item classroom behavior scale, a dose-dependent relationship between lead and outcome was found. Similar results on the rating scale were subsequently reported by Yule et al in England,[14] and Hatzakis et al in Greece.[15]

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10 de la Burde, B; Choate, MS (1975): Early asymptomatic lead exposure and development at school age. J. Pediatr.87, 638-642.

11 Landrigan, PJ: Whitworth, RH: Baloh, RW; Staehling, NW; Barthel, WF; Rosenblum, BF (1975): Neuropsychological dysfunction in children with chronic low level lead absorption. *Lancet*. 1, 708-712.

12 Perino, J; Ernhart, CB (1974): The relation of subclinical lead level to cognitive and sensorimotor impairment in black preschoolers. *J. of Learning Disabilities*.

13 Needleman, HL; Schell, A; Bellinger, D; et al (1990): The long-term effects of exposure to low does of lead in childhood: An 11-year follow-up report. *New Engl J Med*. 311, 83-88.

14 Yule, W; Urbanowicz, MA; Lansdown, R et al (1984): Teachers' ratings of children's behavior in relation to blood lead levels. Brit J of Developmental Psychology. 2, 295-305.

15 Hatzakis, A; Kokkevi, A; Maravelias, C; et al (1989): Psychometric Intelligence Deficits in Lead-exposed Children. In: M.A.Smith, L.D. Grant, A.I. Sors (eds): *Lead Exposure and Child Development an International Assessment*. 1st ed. London, Kluwer Academic Publishers. 211-223.

Although the mean difference in IQ sores was 4 points, a shift of the distributions of this magnitude increases the proportion of children with severe deficits 4-fold, from 4% to 16%.

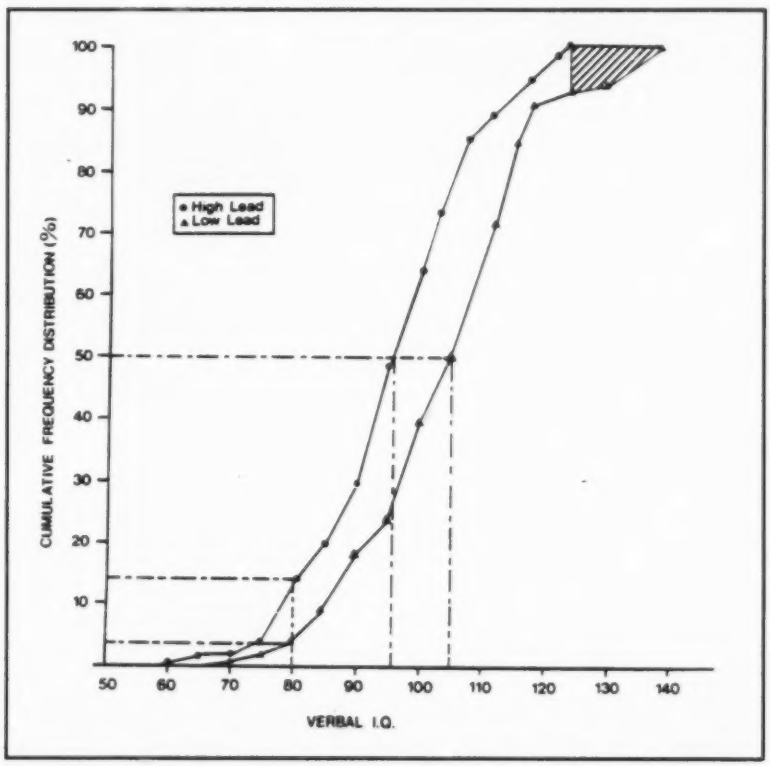

Figure 1 – Cumulative frequency distribution of IQ in high and low lead groups. Note that a shift of 6 points in the median is associated with a 400% increase in the rate of children with IQs below 80, and a truncation of children with IQs above 125. N=2300.

In addition, this shift truncates the top of the distribution, and prevents 5% of the sample from achieving superior function (IQ > 125). The Office of Technology Assessment has estimated the number of American children who were deprived of truly superior function because of this shift in function due to low level lead exposure at 1.3 million.[16]

In the mid 1980s, considerable attention was directed at low level lead exposure in childhood. Among the most noteworthy studies

16 United States Congress. Office of Technology Assessment. Neurotoxicity: Identifying and Controlling Poisons of the Nervous System. OTA-BA-436. Washington, DC, US Government Printing Office.

were Fulton et al,[17] examining middle class children in Edinburgh, Scotland; Hatzakis et al,[15] in Lavrion, Greece, and Hansen et al in Denmark.[18] These investigations showed a deficit in mean IQ in the range of 9-11 points. The exposure levels in the Danish and Scottish studies were considerably lower than those previously reported, with changes observed as low as 10 µg/dl.

Smith et al, studied 400 British school children and after controlling for a number of confounders, reported that the relationship between lead and IQ was not significant.[19] Examination of the dose-response relationship, however, shows a clear decline of IQ with increasing tooth lead. When the data were examined using

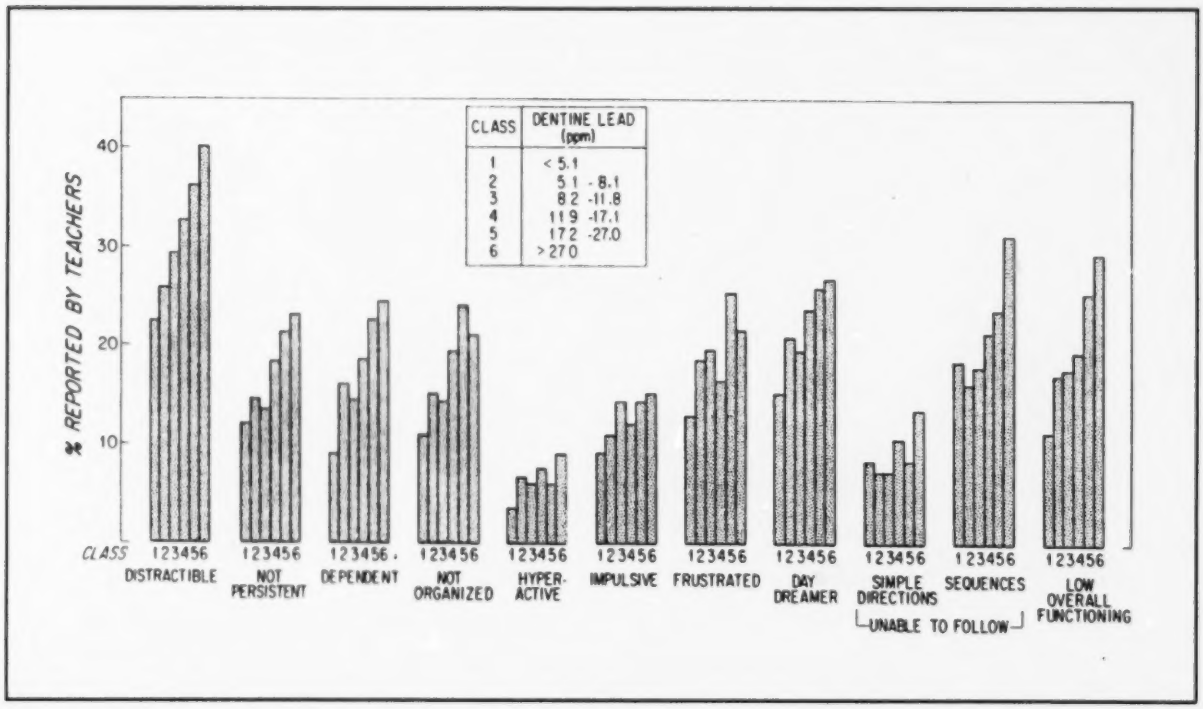

Figure II – Teachers' ratings of children in relation to their dentine lead levels. The proportion of bad ratings in each group is shown. Children are grouped by increasing concentrations of lead in their teeth. N=2146.

17 Fulton, M; Raab, G; Thomson, G; Laxen, D; Hunter, R; Hepburn, W (1987): Influence of blood lead on the ability and attainment of children in Edinburgh. *Lancet*. 1,1221-1226.

18 Hansen, ON, Trillingsgaard, A; Beese, I; et al (1989): A neuropsychological study of children with elevated dentine lead level: Assessment of the effect of lead in different socio-economic groups. *Neurotoxicology and Teratology*. 11,205-213.

19 Smith, MA; Grant, LD; Sors, AI (Eds.)(1989): *Lead Exposure and Child Development an International Assessment*. 1st ed. (Series Eds: Smith, MA; Grant, LD; Sors, AI) Butler and Tanner Lts., London.

multiple regression analysis, a significant relationship between lead and IQ in male subjects was demonstrated.[20]

Although most attention has been paid to the effects of lead on intelligence, lead's impact is not limited to that sphere. It had long been observed, since Byers report at least, that children who had been lead poisoned frequently were overactive and aggressive. There is a growing body of evidence demonstrating lead effects on attention and behavior. Teachers in the 1979 Boston area study, who did not know their pupils' lead levels, reliably reported that children with higher lead levels were more distractible, disorganized, less able to follow directions and were more hyperactive.

Lansdown and colleagues found that lead was associated with higher scores on the overactivity factor of the Rutter B2 scale, and on the conduct problem, inattentive passive, and hyperactive scales of the Connors Questionnaire and the Taylor-Sandberg Hyperactivity Score.[21] Teachers who rated English school children on the same items as the Boston Scale, found similar relationships to those shown in Figure II, Raab et al, studying the same Edinburgh subjects as Fulton, found that lead was related to inspection time, a measure of attention.[22] Thomson,[23] studying the Edinburgh sample, reported that lead was related to hyperactivity and aggressive/antisocial scores on the Rutter Teachers' Scale. Similarly, Fergusson and colleagues showed that lead was significantly related to scores on the inattentive/restlessness items of the Rutter and Connors Scales after controlling for covariates.[24] These reports suggest that attentional function may be a sensitive target for lead. It argues that a portion of children with ADD may have had unrecognized lead toxicity.

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20 Pocock, SJ; Ashby, D; Smith, MA (1987): Lead exposure and children's intellectual performance. *International J of Epidemiology*. 16,57-67.

21 Lansdown, R; Yule, W; Urbanowicz, MA et al (1983): Blood Lead, Intelligence, Attainment and Behavior in School Children: Overview of a Pilot Study. In: Rutter, M; Jones, RR (Eds): *Lead versus Health*. Great Britain, John Wiley & Sons, 267-296.

22 Raab, GM; Thomson, GOB; Boyd, L; Fulton, M; Laxen, DPH (1990): Blood lead levels, reaction time, inspection time and ability in Edinburgh children. *Br. J. Deve. Psychology*. 8, 101-118.

23 Thomson, GOB, Raab, GM; Hepburn, WS; Hunter, R; Fulton, M; Laxen, DPH (1989): Blood lead levels and children's behavior - Results from the Edinburgh lead study. J. Child. Psychol. and Psychia. 30, 515-528.

24 Fergusson, DM; Fergusson, JE; Horwood, LJ; Kinzett, NG (1988): A longitudinal study of dentine lead levels, intelligence, school performance and behavior. Part II Dentine lead and cognitive ability. *J. Child Psychol. and Psychia*. 29, 793-809.

Long Term Consequences of Early Exposure

This question has been ignored until quite recently. We followed our subjects up at two time periods, when they were fifth graders, and when they were young adults. In the fifth grade, lead was significantly associated with increased grade retention, and high lead subjects tended to have lower IQ scores, and greater need for special school services.[25]

As young adults, the results were even more striking. We were able to bring 132 of our original sample of 270 subjects back for testing 12 years later.[26] Their mean age was 18.7 years. Having elevated tooth lead was associated with a number of problems in adulthood. High lead subjects had an adjusted risk of 6.8 of not graduating from high school, and a risk of 5.8 of reading disability. Of the 10 subjects who had a diagnosis of clinical plumbism in 1976, 50% had a reading disorder. and 42% had school failure. Lead exposure was also associated with lower class standing in

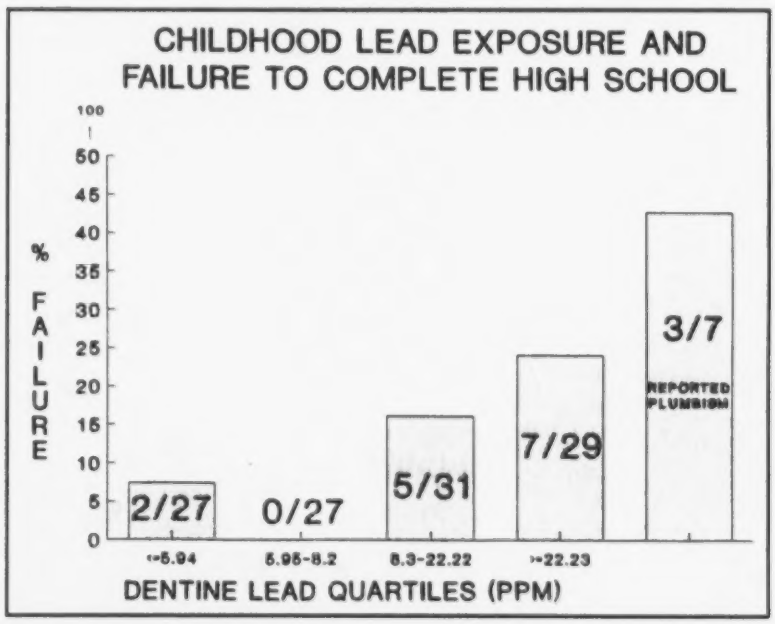

Figure III – High school graduation rates in relation to early dentine lead concentrations. Ten subjects with clinical toxicity were included. Three were still in school at the time data were collected.

25 Bellinger, D; Needleman, HL; Bromfield, R; Mintz, M (1984): A follow-up study of the academic attainment and classroom behavior of children with elevated dentine lead levels. *Biol. Trace Elem. Res.* 6, 207-223.

26 Needleman, HL; Schell, A; Bellinger, D; Leviton, A; Allred, EN (1990): The long-term effects of exposure to low doses of lead in childhood: An 11-year follow-up report. *New Engl. J. of Med.* 322, 83-88.

the final year of high school, with increased absenteeism, lower vocabulary scores, and impaired fine motor function. Lead exposure at these doses seems to produce permanent effects that influence academic adjustment. Byers' question about the relationship between lead and school adjustment was prescient.

Table 1 – Regression of Outcomes in Young Adulthood on Dentin Lead Levels in Childhood.*

	Multiple Regression	
Outcome Variable	Parameter Estimate	P Value
Highest grade achieved	–0.027	0.013
Reading grade equivalent	–0.072	0.001
Class standing	–0.006	0.048
Absence from school	4.73	0.010
Grammatical reasoning	0.178	0.011
Vocabulary	–0.122	0.001
Finger tapping	–0.133	0.010
Hand-eye coordination	0.048	0.010
Reaction time		
Preferred hand	12.9	0.042
Nonpreferred hand	10.3	0.060
Minor antisocial behavior	–0.739	0.038

* The following covariates were controlled for in the multiple regression analysis: age, sex, birth order, family size, mother's age at the subject's birth, length of the neonatal stay in the hospital, mother's education level, mother's IQ, socioeconomic status, and current alcohol use.

Prenatal Exposure to Lead

Lead crosses the placenta, and can be measured in the umbilical cord blood.[27] The impact of lead exposure on the fetal brain when it is laying down connective fibers is an important consideration. We studied the effects of exposure in a cohort of 12,000 subjects enrolled at the Boston Hospital for Women. Umbilical cord blood lead levels declined approximately 10% per year.[28] This was

27 Scanlon, J (1971): Umbilical cord blood lead concentration: Relationship to urban or suburban residency during gestation. *Amer. J. Dis. Child.* 121, 325-326.

28 Rabinowitz, MB, Needleman, HL (1982): Temporal Trends in the Lead

closely correlated with the reduction of lead in gasoline. We first examined the relationship between umbilical cord lead and neonatal status in 5000 births.[29] Controlling for a number of factors, lead was significantly associated with the rate of minor malformations. In this study, lead was not associated with birth weight, major malformations, infections or jaundice. Others have reported no relationship between cord blood lead and malformations, but their sample sizes were too small to permit that assertion.[30] Other investigations have found that lead exposure is related to intrauterine growth and growth during the first year of life.[31]

We followed 249 children from this group forward in time at 1, 6, 12, 24, 57 month intervals, and again at 10 years of age. At 24 months, umbilical cord blood lead was significantly related to Bayley Developmental Scales, after adjustment.[32] At 57 months of age, the prenatal exposure was no longer influential, except in one group of subjects: infants of low SES who had postnatal exposure. But the effect of 24 month blood lead levels on intelligence now became significantly related to 57 month performance. The high lead group had a decrease in IQ of 8 points compared to the low lead group. It is notable that the lower boundary of the high lead group was 10 µg/dl. Other investigators who found an effect of early exposure on outcome were Dietrich[33] and McMichael and colleagues.[34]

Concentrations of Umbilical Cord Blood. *Science*. 216, 1429-1431.

29 Needleman, HL; Rabinowitz, M; Leviton, A; Linn, S; Schoenbaum, S (1984): The relationship between prenatal exposure to lead and congenital anomalies. *JAMA*. 25, 2956-2959.

30 Ernhart, CB; Wolf, AW; Kennard, JJ et al (1986): Intrauterine exposure to lead: The status of the neonate. *Arch Environ Health*. 41, 287-291.

31 Shukla, R; Bornschein, RL; Dietrich, KN et al (1987): Effects of fetal lead and early postnatal lead exposure on child's growth and stature: The Cincinnati lead study. In: Lindberg, S; Hutchinson, T (Eds.) *Sixth International Conference on Heavy Metals in the Environment*. Edinburgh, CEP Consultants. 210-215.

32 Bellinger, D; Leviton, A; Waternaux, C; Needleman, HL; Rabinowitz, M (1987): Longitudinal analyses of prenatal and postnatal lead exposure and early cognitive development. *New Engl. J. Med*. 316, 1037-1043.

33 Dietrich, K; Krafft, K; Beir, M; Bornschein, R (1987): Early effects of fetal lead exposure: Developmental findings at six months. In: *Heavy Metals in the Environment: International Conference*, New Orleans (Eds.) Lindberg, S; Hutchinson, T) CEP Consultants, Edinburgh, 63.

34 McMichael, AJ; Baghurst, PA; Wigg, NR; Vimpani, GV; Robertson, EF; Roberts, RJ (1988): Port Pirie Cohort Study: Environmental exposure to lead and children's abilities at the age of four years. *New Engl. J. of Med*. 319, 468-47.

Synthesis of Studies

The question of lead's impact on IQ at lesser doses may have engendered more controversy than any other issue in environmental health. There are a number of reasons for this. The effects of lead are nonspecific. Many factors can lower IQ scores, increase restlessness and distractibility, and impair speech development. Many of these, such as undernutrition, iron deficiency, infections, head injury, and inferior prenatal care, are more frequent in the poor. This confounding makes the effects of lead more difficult to isolate. It offers, to those who would dismiss the hazards of lead, a series of alternate explanations.

Not all studies have shown an effect, and some argue that this is an inconsistency and that it means there is no effect in nature. The controversy is exacerbated by interests who do not want to spend the money to remove lead from the environment. Pressure issues from the real estate industry, the lead additive industry, and others.

Simply tallying the number of studies that find an effect and those that do not is a poor way to synthesize studies. A relatively new method of combining studies is metaanalysis. In this method, each study is treated as a data point in a larger metastudy, and statistical techniques used to synthesize the data are applied.

A recent metaanalysis of all 24 informative modern studies handled those using blood separately from those using teeth.[35] A separate and more detailed analysis was done for the 12 studies that contained enough information to estimate effect size, expressed as partial r. The partial r's ranged from –.27 to -.003. The joint P value for all blood lead studies was .0001. For the tooth studies the P value was .0005. When all 24 studies were included in the analysis, the P value was < .0001. We then investigated whether this observation could be due to the differential publication of studies that showed a positive effect. To accomplish this, a "file drawer" analysis was done. This procedure estimates how many unpublished no-effect studies would be required to vitiate the observed P value. To bring the P value for this study to non-significance, there would have to be 93 unpublished studies that showed no lead effect in file drawers around the world. The conclusion to be drawn is that there is strong convergence in modern studies that low dose lead is neurotoxic. Since this paper was published, there have been at least 5 other reports showing an effect of lead on IQ.

35 Needleman, HL; Gatsonis, C (1990): Low level lead exposure and the IQ of children. *JAMA*. 263(5),673-678.

What is the Toxic Dose of Lead?

The defined toxic dose of lead has decreased over time as studies with larger samples, more sensitive measures of outcome and better statistical techniques have been reported. In the 1960s, the toxic level was defined as 60 µg/dl. This was revised downward by DHEW in 1972 on the basis of available information to 40 µg/dl. The Centers for Disease Control (CDC) reevaluated the literature in 1978, and lowered the threshold to 30 µg/dl, and in 1985 to 25 µg/dl. A thorough reevaluation was undertaken by CDC in 1991. The CDC report concluded:

> *New data indicate significant adverse effects of lead exposure in children at blood lead levels previously believed to be safe. Some adverse health effects have been documented at blood lead levels at least as low as 10 micrograms per deciliter of whole blood (µg/dl)...*
> *The 1985 intervention level of 25 µg/dl is, therefore, being revised downwards to 10 µg/dl.*

This definition was not achieved without a considerable amount of thought and debate. A number of studies in children show IQ effects at this level. Animal studies in which the dose is precisely controlled, and blood lead levels determined over the entire history of the subjects' lives show effects in this range.[36] Both EPA and the Agency for Toxic Substances and Disease Registry have stated that the No Effect Level may in fact be lower.

What is the Extent of Excess Lead Exposure?

The most current data on blood lead levels derives from the National Health and Examination Survey of 1976-1980. This is a balanced probability sample of 26,000 American adults and children. A blood lead level was obtained from approximately 5,000 children from 9 months to 6 years of age. Since the time this survey was conducted, blood lead levels in this country have declined. A good portion of this decline can be assigned to the removal of lead from gasoline over the span of the study.

Mushak and Croccetti reviewed the epidemiology of lead for the Report to Congress of the Agency for Toxic Substances and Disease Registry,[2] and adjusted the prevalences for the secular decline of blood lead concentrations, and estimated the contemporary blood lead concentrations.

36 Rice, DC (1984): Behavioral deficits (Delayed matching to sample) in monkeys exposed from birth to low levels of lead. *Toxicol Appl Pharmacol.* 73,237-245.

They state:

> *In short, about 2.4 million white and black children (ages 6 months to 5 years) or about 17% of such children in U.S. SMSAs, are exposed to environmental sources of lead at concentrations that place them at risk of adverse health effects. This number approaches 3 million black and white children if extended to the entire U.S. child population.* ***If the remaining racial categories are included in these totals, between 3 and 4 million U.S. children may be affected.***

Why Has Progress In Lead Control Been Slow?

It is pertinent to ask why, in the face of an extraordinary amount of convergent information on the toxicity of lead at low dose, and on the epidemiology of lead in children, the response of society and government has been so hesitant. There are a number of reasons that warrant examination.

Not all studies reported an effect, and this question has become one of the most contentious issues in toxicology. The conclusion as to whether lower, asymptomatic levels of exposure are neurotoxic, has strong implications for regulating lead in gasoline and other sources. The financial and hygienic stakes are high, and the debate is often heated.

Lead produces no characteristic picture, and the effects at lesser doses is not dramatic. If thalidomide, when given to mothers, had resulted in lower IQ scores and attention deficits in offspring, it would still be administered during pregnancy. Lead exposure has been traditionally considered a disease of poor minority children. Associated with this belief is the conviction that if the mother had cared for the child more assiduously, the problem would not have happened. Once the burden has shifted to the parents, medical and public health responsibility can rest. If lead poisoning were a disease that preferentially sought out middle class children, it would probably have been eradicated by now.

Academic pediatrics has to a considerable extent ignored the problem of low level lead exposure. There are a number of excellent pediatric departments that give little attention to teaching about low dose lead toxicity, and some that do not screen for lead in their outpatient clinics. As a result, their students and house officers do not see any cases of lead toxicity, and their belief that the disease is rare is confirmed. Nor have educators generally realized the import of lead exposure for the academic success of their students.

The Department of Housing and Urban Development has not fulfilled its major responsibility in dealing with lead paint in either its public housing or in private housing under its aegis.

The lead industry, through its trade organizations and academic consultants, has worked hard at camouflaging the effects of lead at low dose. This is not a new activity. When Byers published his paper in 1943, he was threatened with a million-dollar law suit. More recently, a claim that I was guilty of scientific misconduct was placed by individuals supported by the industry. This charge was unanimously rejected by the University Hearing Board.

There is a widely held misperception that since lead has been removed from gasoline, and a ban on lead in household paint has been in place since 1976, the problem has been taken care of. In fact, there are over 3 million homes in which children live that have deteriorated leaded surfaces. Almost every child in one of these houses has an elevated lead level. Curiously, coexistent with the belief that the problem has been eradicated is the belief that the problem is too big for our society to handle in a time of budgetary deficits.

All of the above factors explain some of the reasons for the lack of a serious penetration into this problem. Unlike the AIDS virus, lead is no mystery ; we know enough about the agent, its whereabouts, and what needs to be done to make a strong preventive effort plausible and likely to succeed.

Implications of Lead Toxicity for Educators, Legislators and Child Development Specialists

The prevalence data show that lead exposure is extraordinarily high, and that it affects children of all socioeconomic strata and race. But as is true of many of the assaults of poverty, the poor and minorities are more heavily exposed. Lead exposure is more frequent in those groups where subnormal intelligence, attention disorders, and behavior problems are found at higher rates. This is not to say that lead is the only cause of these problems. Most studies of children in these populations do not measure lead burden, and enter it into their models.

It is important to remember that over half of the black children in poverty have elevated blood lead levels. Cowan and Leviton, using our data from the Boston Study, estimated the attributable risk for low overall classroom performance in that group.[37] They

37 Cowan, L; Leviton, A (1980): Epidemiologic considerations in the study of the sequelae of low level lead exposure. In: *Low Level Lead Exposure: The Clinical Implications of Current Research.* (Ed.) Needleman, HL. Raven Press, NY. 91-120.

used the Teachers' rating scale, and compared rates on the question: "Is this child's performance on a par with his or her classmates?"

Attributable Risk $=(I_e-I_o)/I_e$

where I_e= incidence of "No" answers in exposed group
and I_o= incidence in unexposed group

A.R. = (.26-.08)/.08=.69

Sixty-nine percent of low overall classroom functioning in children with high dentine lead levels was attributed to their lead exposure.

More to the point is the population attributable risk, that portion of the population of school children with low function attributable to lead.

Population Attributable Risk $=(I_p-I_o)/I_p$

where I_p=Incidence in the population
and I_o=Incidence in the unexposed group

P.A.R.= (.14-.08)/.14= .43

In this study, 43% of low overall function in the total school population would be attributed to elevated lead exposure.

The relationship of lead to altered reaction time, scores of Teachers' and Parents' ratings cited earlier demonstrate that lead is associated with attentional problems and in some studies, aggressive behavior. If a male child has attention deficit disorder and one sign of conduct disorder, he has a .60 risk of appearing on a police blotter multiple times by the time he is 18.[38] The attributable risk for attentional dysfunction given elevated lead is .5. By multiplying the lower boundary of the 95% confidence limits for the two contingencies, we can estimate the joint probability for delinquent behavior, given elevated lead level.

$P_{del/lead}= P_{del/add} \times P_{add/lead} = .4 \times .5 = .2$

A reasonable estimate of the attributable risk for delinquency in high lead children is 20%. We are currently studying this question.

Denno[39] analyzed the data from the Perinatal Collaborative Study of nearly 1000 Philadelphia subjects followed from birth

38 Magnusson, D; Stottin; Dunner, A (1983): Agression and criminality in a longitudinal perspective. In: *Antecedents of Agression and Antisocial* Behavior. (Eds.) Van Dusen, KT and Mednick, SA. Kluwer-Nijhoff, Boston.

through early adulthood, and obtained police records for juveniles and adults. In males, lead poisoning was one of the strongest predictors of number and severity of delinquent offenses. The data from the Boston followup study, showing increased rates of school failure, reading deficits, and lower vocabulary scores in lead exposed subjects, add to the credibility of this observation. If a male drops out of school, and cannot read well, there are few vocational opportunities beckoning to him, and the path to antisocial behavior widens.

Steps Toward Primary Prevention

In the winter of 1990, Dr James Mason, Assistant Secretary of Health, took an historic step. He directed Dr. Vernon Houk, Director of the Center for Environmental Health and Injury Control of the Centers for Disease Control to draft a plan to eradicate childhood lead poisoning. Six months later, that plan was on his desk, and on February 21 1991, it was announced.[1] This represented a fundamental shift in the Public Health Service's response to the modern picture of lead toxicity in the US.

Instead of finding cases and treating them, the plan called for a staged attack on the problem, consisting of increased childhood lead poisoning prevention programs and activities, effective abatement of leaded homes, reduction of lead from other sources, and a national surveillance effort. Perhaps the most important part of this historic document is the cost benefit analysis that CDC commissioned. The economists monetized the costs for health care, special education, lowered earning rates, and abatement. From these data, they estimated that the cost for deleading a modest pre 1950 house at $2225. They estimated the monetized benefits from decreased medical and special educational costs, and from increased wages associated with higher IQs at $4323 per abated unit. The discounted costs for abating all pre-1950 houses was $33 billion. The discounted benefit was $62 billion. CDC's economists tested various models and found that the benefits were robust to changes in assumptions of the costs of abatement, efficacy of abatement and the discount rate. Because metrics for good health and better living quality are imprecise, while metrics for labor and material costs are precise, cost benefit analyses generally underestimate the benefit of environmental control. Nevertheless, this analysis provides a sound basis on which to undertake the program. Another unmeasured benefit is the lift to communities that accompanies attention to the quality of the residents' lives, and perhaps most important, the possibility that jobs would be created in these areas.

39 Denno, DW (1990): *Biology and Violence from Birth to Adulthood*. Cambridge University Press, Cambridge, New York.

Ending Childhood Lead Poisoning

Lead poisoning, like auto accidents, violent deaths, and lung cancer is primarily due to conscious choices in human activity. This should be a source of some optimism. Being manmade, lead poisoning is potentially completely preventable. A common response is that this notion is a utopian delusion. After all, aren't the states and local authorities impoverished? Is not our deficit in the hundreds of billions? Where could the money be found to delead 30 million houses?

We are immersed in a wave of unrecognized chronic pessimism about our ability to exert any control over our future. Some economists argue cogently that spending on physical repair of our infrastructure contributes to wealth and thus will eventually reduce our national debt. The same applies to removing lead from housing stock, which can provide approximately half a million jobs for the next 10 years. And investing in improving human capital — the brains of millions of our children — could be one of the best ways of enriching our future.

The places where lead has been accumulated in superabundance is precisely the places where there are two dire shortages: jobs and decent housing. One way to deal with this immoral and dangerous disequilibrium would be to train unemployed residents of these neighborhoods in proper and safe deleading, and pay them. The same health dollar would decrease unemployment, improve housing, and wipe out lead paint poisoning.

The prospect of driving a disease rate down to zero is not new. In 1963, the Secretary General of the World Health Organization was approached with a proposal to eradicate small pox in the world. He thought this was a utopian vision, and reasoned that since it would fail, it would be best if an American was chosen to lead the project. Thus Dr. Donald Henderson was appointed to direct the smallpox campaign. In 1977 the last case of smallpox was reported. Lead poisoning can have the same history given the same vision, commitment and quality of leadership.

National Health/Education Consortium

Good health is a significant determinant of a child's ability to learn and succeed in school. The health and education sectors, however, have historically approached programs and services for children from different perspectives. Recognizing the need for better integration of health and education programs for children, the National Commission to Prevent Infant Mortality and the Institute for Educational Leadership organized the NATIONAL HEALTH/EDUCATION CONSORTIUM in May 1990.

The project is unique in that it has brought together leaders from 56 national health and education organizations, representing nearly 11 million constituents, to bridge the gap between the worlds of health and education and to generate unified action for children. Promoting the full potential of children and providing them with the best opportunities for success will require changes in the systems which currently provide health and education services. Reforms are needed to develop more collaborative and cohesive policies and programs, unify agencies and funding streams, and provide a more comprehensive approach to children's problems.

Toward this end, the NATIONAL HEALTH/EDUCATION CONSORTIUM's activities focus on three major goals: to improve public policy in addressing the need for a better coordinated health and education delivery system; to strengthen communication and dissemination of information between health and education activities and policymakers; and to identify exemplary program models and practices which more effectively integrate health and education services. The Consortium involves educators, health professionals, policymakers, administrators, civic leaders, advocates, and parents in its efforts to bring together the health and education communities in a more integrated fashion.

The Consortium is able to attract high profile business, political, health and education leaders to give visibility and implement its mission at the federal, state, and local levels. It does not represent any particular special interest group, but can bring to bear the weight of national association consortium members on behalf of its mission. In addition, the consortium creates state and local networks across the country that will plant the seeds for similar collaborative efforts at those levels.

The foundation of the Consortium's efforts can be found in the report, *Crossing the Boundaries between Health and Education*, which documents clinical research and programs that exemplify the relationship between children's health and their learning potential. To complement this report, the Consortium is releasing a series of papers which focus on various topics relating to health and education.

The NATIONAL HEALTH/EDUCATION CONSORTIUM is supported by The Prudential Foundation, the Honeywell Foundation, the AT&T Foundation, Metropolitan Life Insurance Company, and the Procter & Gamble Fund. Additional support has been provided by the U.S. Department of Health and Human Services, and the U.S. Department of Education.

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National Health/Education Consortium Members and Profiles

American Academy of Family Physicians: 66,000 physicians

American Academy of Pediatrics: 39,000 physicians

American Association of Colleges for Teacher Education: represents 700 member institutions teacher education programs

American Association of School Administrators: 18,517 school administrators

American College of Nurse-Midwives: 3,000 certified nurse-midwives

American College of Obstetricians and Gynecologists: 29,848 obstetricians and gynecologists

American Dental Association: 138,217 dentists

American Federation of Teachers: 750,000 teachers, para-professionals (teacher aides), school-related personnel, healthcare workers, federal and state employees

American Hospital Association (MCH Section): 5,870 hospitals and physicians

American Indian Health Care Association: represents 36 programs and clinics which focus on the health care of American Indians

American Medical Association: 300,000 physicians

American Nurses Association: 201,000 registered nurses

American Public Health Association: 30,977 physicians, nurses, therapists, health technicians, health support personnel, and other health professionals

American School Health Association: 3,000 health educations, nurses, physicians, and dieticians

American Speech-Language-Hearing Association: 67,393 speech-language pathologists, audiologists, scientists, researchers, and educators

Association for the Care of Children's Health: 4,200 nurses, child life workers, and parent leaders

Association for Supervision and Curriculum Development: 153,000 teachers, school administrators, college professors, and school board members

Association of American Medical Colleges: 126 U.S. medical schools, 450 teaching hospitals, and 92 academic professional societies

Association of Maternal and Child Health Programs: represents 269 maternal and child health programs and their directors

Association of Schools of Public Health: represents 13,000 deans, faculty, and students of schools of public health

Association of State and Territorial Dental Directors: represents 58 state and territorial dental directors.

Association of State and Territorial Health Officials: represents the 58 health officers from each of the United States and its territories.

Council of Chief State School Officers: represents 56 public officials who head departments of elementary and secondary education in each state and extrastate jurisdiction

The Council of Great City Schools: represents 46 of the largest urban public school districts in the United States

The Elementary School Center: represents 500 child-serving professionals

Healthy Mothers, Healthy Babies Coalition: represents 95 non-profit health education groups, and state and local education groups

NAACOG (The Organization for Obstetric, Gynecologic and Neonatal Nurses): 24,000 nurses

National Alliance of Black School Educators: 3,000 African-American teachers for Grades K-12

National Association for Asian and Pacific American Education: 594 members representing administrators, teachers, institutional aids, social workers, mental health workers, and students among others

National Association for the Education of Young Children: 77,000 members representing a wide range of early childhood professionals

National Association for Partners in Education: 5,5000 volunteers, presidents and executives of private businesses, teachers, and administrators

National Association of Children's Hospitals and Related Institutions: represents 108 hospitals

National Association of Community Health Centers: represents 600 health care facilities

National Association of Elementary School Principals: 36,000 elementary school principals, middle school principals, school superintendents, teachers, professors, and instructors

National Association of Hispanic Nurses: 1,000 Hispanic nurses

National Association of Pediatric Nurse Associates and Practitioners: 2,800 pediatric nurse associates and practitioners

National Association of School Nurses, Inc.: 5,800 school nurses

National Association of Secondary School Principals: 41,000 secondary school principals, administrators, guidance counselors, activities directors, and college professors

National Association of Social Workers, Inc.: 137,763 members in all fields of social work

National Association of State Boards of Education: represents 600 state boards of education and their members

National Association of WIC Directors: 624 state directors, nutrition coordinators, and local agency directors

National Black Nurses Association: 7,000 African-American nurses

National Coalition of Hispanic Health and Human Services Organizations (COSSMHO): 700 organizations serving the Hispanic population, representing Hispanic physicians, nurses, and students

National Community Education Association: 1,600 teachers, superintendents, administrators, community education directors and coordinators, faculty and administrators of teacher education institutions and programs, community activists, private businesses, and state administrators

The National Congress of Parents and Teachers: 6.8 million parents, K-12 classrooms teachers, principals, school administrators, and students

National Education Association: 2 million K-12 classroom teachers, professors, educational support personnel, and students

National Head Start Association: 150 nationwide agency members and 30 individual members

National Indian Education Association: 2,500 members, most of whom are Native American educators

National Medical Association: 16,000 minority physicians

National Mental Health Association: 550 local affiliate mental health associations representing mental health care providers, clients, and community health care centers

National Perinatal Association: 6,000 physicians, nurses, nurse-midwives, social workers, and consumers of perinatal services

National Rural Health Association: represents 1,750 community, migrant, and homeless health centers and their staffs

National School Boards Association: represents 52 state school board associations

National School Public Relations Association: 2,200 teachers, principals, administrators, retired teachers, students, and public relations personnel

Society for Neuroscience: represents 18,000 neuroscientists

Zero to Three – National Center for Clinical Infant Programs: represents 7,500 programs for high risk children and families, as well as individuals

This document does not necessarily reflect the viewpoints or positions of any of the Consortium's members.

Governor Lawton Chiles (FL)
Chairman
National Commission to Prevent Infant Mortality

NATIONAL
HEALTH/EDUCATION
CONSORTIUM

William S. Woodside
Chairman of the Board
Institute for Educational Leadership

November 17,1992

TO: Interested Media
FROM: Rae K. Grad, Executive Director, National Commission to Prevent Infant Mortality
Mike Usdan, President, Institute for Educational Leadership
RE: The Poisoning of America's Children: Lead Exposure, Children's Brains and the Ability to Learn

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We are pleased to provide you with a copy of our most recent publication from the **National Health/Education Consortium**, The Poisoning of America's Children: Lead Exposure, Children's Brains and the Ability to Learn.

Herbert L. Needleman, Professor of Psychiatry and Pediatrics at the University of Pittsburgh and author of the report, is a leader in the field of lead exposure. His expertise has allowed him to conduct extended studies on children to track the long term effects of low level lead exposure. His findings show that lead not only affects children's abilities to learn, but affects their behavior patterns as well.

In recent weeks, new studies on the effects of lead on a child's ability to learn have been in the forefront of the news. The United States government has recently taken action to reduce lead exposure, including lowering the level of lead considered safe in the blood, and requiring lead screening as part of routine physical examinations. Unfortunately, this is not enough.

Despite years of concern about the toxic effects of high-lead exposure and recent knowledge about the less apparent low-dose effects, it is estimated that between three and four million children in the United States are still being exposed to concentrations of lead so dangerous, the cognitive and social development of these children could be compromised.

This paper addresses many lead exposure questions including the long term consequences of early exposure, prenatal exposure to lead, what is considered a toxic dose of lead, and why so little is being done to control lead exposure. The uniqueness of this report is that it discusses long term effects of lead exposure, including the behavioral problems that may cause school failure that are often faced by children with low-level lead exposure.

The National Health/Education Consortium is a joint initiative of the National Commission to Prevent Infant Mortality and the Institute for Educational Leadership. Comprised of leaders from 56 national health and education organizations, representing nearly 11 million constituents, the Consortium strives to bridge the all too often separate worlds of health and education into unified action for children.

We hope this report will contribute to your coverage of health issues, especially the effects of lead, how it is poisoning our children, and what we need to do as a society to eradicate it from our lives.

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